

POSTOPERATIVE ENTIRE LEFT LUNG COLLAPSE BY MUCUS PLUGS

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Introduction

Postoperative lung complications (PPC) may occur in up to 20% in emergency surgeries. We present a case of 73 year old male who presented with an entire left lung collapse in the postoperative period after repair of duodenal perforation despite adequate analgesia, aggressive chest physiotherapy and deep breathing exercises. Using fiberoptic bronchoscopy, the mucus plugs occluding both the upper and lower lobes of the left lung were removed. The collapsed lung was then successfully re-expanded with oxygen insufflation during bronchoscopy and thereafter with recruitment manoeuvres. Relevant chest X-rays and bronchoscopy images are also presented.

Case Report

A 73 year old male was admitted with severe epigastric pain. His medical history was unremarkable except that he was a heavy smoker (50 pack years). He had been smoking until the previous day of hospital admission. On examination, he was moderately dehydrated, tachycardic (110/min) and tachypnic (20/min). His room air saturation (SpO₂) was 78% which improved to 92-94% on oxygen supplementation (6 L/min) by face mask. Clinical examination of chest revealed decreased bilateral basal air entry, normal vesicular breath sounds and no added sounds. Abdomen was rigid and tender. The clinical impression was hollow viscous perforation with resultant peritonitis, and a decision for exploratory laparotomy was made. The

bedside preoperative chest X-ray was suggestive of chronic obstructive pulmonary disease (COPD), and a few bullae noted bilaterally in the upper zones.

Given the emergency nature of the procedure, no formal pulmonary function testing or preoperative arterial blood gas analysis were done. Also, both these would not have changed the immediate peri-operative care of the patient. The bedside clinical examination (tachypnea - 20/min, decreased bilateral basal air entry, with normal vesicular breath sounds and no added sounds, room air SpO₂- 78%) and presence of bilateral bullae in the upper zones on chest X-ray were consistent with acute on chronic respiratory failure. However, prior to induction of anaesthesia the patient received inhaled bronchodilators (salbutamol 5mg + Ipratropium 0.5mg) to treat any possible reversible bronchospasm. The arterial blood gas done during the intra-operative period at an FiO₂ 0.5, PEEP-8, V_t- 550 ml, f- 14, I;E- 1:2, PIP- 20 to 25 cm H₂O showed no evidence of significant chronic CO₂ retention. (pH- 7.34, PO₂- 78, PCO₂- 45, HCO₃⁻ 19, Base Excess- -5).

On laparotomy, a perforated duodenal ulcer in the D2 region was found which was repaired with indirect omentoplasty. During the intra-operative period, the patient remained haemodynamically stable and maintained SpO₂ >95% on intermittent positive pressure ventilation with a FiO₂ of 0.5. Postoperatively, the patient was transferred to ICU and remained on ventilator for about 5 hours. He was gradually weaned off and extubated. In the immediate postoperative period he was comfortable and maintained SpO₂ >95% on oxygen supplementation (6 L/min) by face mask. In view of his COPD, he was advised postoperative aggressive chest physiotherapy and transferred to the ward.

After weaning from ventilator and on transfer to ward on the first postoperative day, the patient received humidified oxygen and aggressive chest physiotherapy (deep breathing exercises, induced cough and chest percussions). Despite this on the third postoperative day, he was again referred to the ICU in view of shortness of breath and desaturation (80%). On examination, he was tachypneic (25/min) with a tracheal deviation to the left, dull percussion note and decreased air entry over left lower chest. The portable supine chest X-ray showed tracheal and mediastinal shift to the left, crowding of ribs and an underlying opacity that was suggestive of left lower lobe collapse. Patient was put on non-invasive ventilation (IPAP: 12-15 cm H₂O, EPAP: 8 cm H₂O, FiO₂- 0.5) and aggressive chest physiotherapy and inhalational bronchodilators were continued. Despite improvement in the level of patient comfort and SpO₂ (92-95%), the Chest X-ray done on the next day showed further radiological deterioration (Figure-

1). A decision to do a bronchoscopy and pulmonary toilet was made as the clinical and radiological pictures were highly suggestive of a mucus plug occluding major airways. The patient was intubated and subsequent bronchoscopy showed two mucus plugs occluding both the upper and lower lobe bronchi of the left lung (Figure-2). They were removed during the procedure and the collapsed lungs re-expanded by oxygen insufflation after wedging the bronchoscope peripherally, and thereafter by recruitment manoeuvres. After overnight ventilation the patient was extubated and the repeat chest X-ray confirmed re-expansion of the collapsed lung (Figure-3).

Discussion

Postoperative lung complications (PPC) occur in 3-4% after elective surgery, and up to 20% in emergency operations¹. The mechanisms postulated to cause atelectasis and airway closure are the loss of respiratory muscle tone and gas resorption during anaesthesia. Avoiding of high inspired oxygen fractions during both induction and maintenance of anaesthesia, intermittent 'vital capacity' manoeuvres and application of PEEP especially in obese individuals may help in lung recruitment and prevent postoperative atelectasis. The incidence of postoperative atelectasis is much higher in the patients undergoing thoracic surgical procedures. This maybe due to a weak cough and the inability to clear bronchial secretions, often a consequence of inadequate analgesia, thoracic muscle injury, chest wall instability or diaphragmatic dysfunction. In addition, patients with lung diseases are prone to increased secretions. The reported risk factors for PPC are age more than 65 years, history of smoking or chronic obstructive airway disease, history of cancer, impaired preoperative cognition, presence of perioperative nasogastric tube, body mass index > 27, surgical incision site involving the upper abdomen, duration of anaesthesia greater than 2.5 hours, and a positive cough test^{2,3,4}. Preoperative blood gases (PaO₂ < 9.33 kPa) and moderate-to-severe airway obstruction on spirometry may also help in predicting PPC⁵.

Our patient was at a high risk of PPC in view of his old age, history of smoking, evidence of chronic obstructive airway disease on the preoperative chest X-ray and upper abdominal incision. So, the patient was given a regime of adequate analgesia, aggressive chest physiotherapy and deep breathing exercises. Also, encouraged to cough and mobilize secretions. Despite these measures he developed left lower lobe collapse on third postoperative day. Due to worsening breathlessness he was transferred to ICU for non-invasive ventilation and other measures to facilitate pulmonary toileting. His Chest X-ray on the next day showed opacification of the entire left hemithorax, proximal air bronchogram, crowding of ribs, mediastinal shift to the

left side and obscuring of diaphragm and heart borders (Figure-1). This was highly suggestive of collapse of the entire left lung due to mucus plugging⁶ and was confirmed by flexible bronchoscopy which revealed two mucus plugs occluding both the upper and lower lobe bronchi of the left lung (Figure-2). Previous studies have also reported the necessity of fiberoptic bronchoscopy to remove proximally located mucus plugs^{7,8}. Interestingly, the only prospective study comparing the efficacy of bronchoscopy with chest physiotherapy in acute lobar atelectasis did not show any difference in outcome at the end of a 24 hour trial period⁹. However, Kreider and Lipson in a recent review conclude bronchoscopy to be the treatment of choice in lobar or segmental atelectasis, though less effective in subsegmental and distal atelectasis¹⁰. Our case also suggests that proximally located large mucus plugs causing lobar atelectasis will need bronchoscopy to remove them and facilitate lung expansion. Air or oxygen insufflation during bronchoscopy helps to re-expand the collapsed lung. This may be done after wedging the scope to the atelectatic regions or using a balloon cuffed flexible bronchoscope or even a Swan-Ganz catheter through the bronchoscope^{11,12,13}.

In conclusion, preoperative and postoperative chest physiotherapy and incentive spirometry help decrease the PPC in at risk patients. However, lobar atelectasis often requires bronchoscopy to remove proximally located mucous plugs and facilitate lung re-expansion.

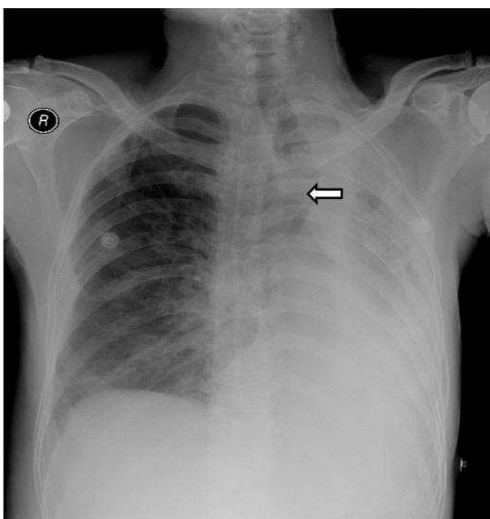


Figure-1. Chest X-ray on the fourth postoperative day- opacification of the entire left hemithorax with an ipsilateral mediastinal shift (arrow), proximal air bronchogram, crowding of ribs, and obscuring of diaphragm and heart borders were highly suggestive of lung collapse due to mucus plugs.

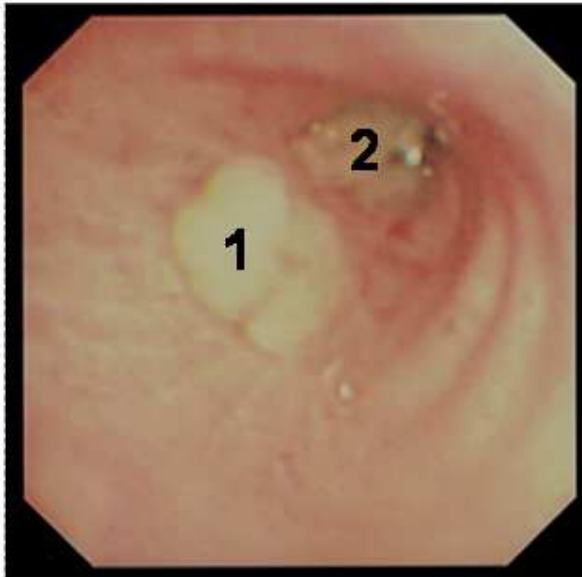


Figure-2. Flexible bronchoscopy confirms two mucus plugs occluding both the upper and lower lobe bronchi of the left lung



Figure-3. Re-expanded lung after bronchoscopy and recruitment maneuvers

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